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Intensive Care and Management of a Suspected Case of Amniotic Fluid Embolism - A Case Report

Dr. Brahmacharimayum Diparani Devi[1], Dr. Sonia Nahakpam[1], Dr. Longjam Eshori[2], Dr. M. Binarani[3]

[1]Post graduate trainee, Regional institute of Medical Sciences, Imphal, India

[2]Senior Resident Doctor, Regional Institute of Medical Sciences, Imphal, India

[3]Associate Professor, Regional Institute of Medical Sciences, Imphal, India

[4]Assisstant Professor, Regional Institute of Medical Sciences, Imphal, India

Abstract: ***The disastrous entry of amniotic fluid into maternal circulation leads to dramatic sequelae of clinical events referred to as Amniotic fluid embolism which has grave maternal and fetal consequences. The diagnosis of AFE is usually made clinically and by exclusion of other causes. The cornerstone of management is a multidisciplinary approach which includes supportive treatment of failing organ systems with which is associated with high mortality.***

Keywords: Amniotic fluid, embolism, diagnosis, management, mortality

1. Introduction

Amniotic fluid embolism is a rare obstetric complication typically occurring during labor, delivery or immediately postpartum that occurs as a result of entry of amniotic fluid and particulate matter into maternal circulation leading to sequelae of this syndrome.

It was first reported in 1926 Meyer.1, [3]Incidence has been reported to range from1 in 800 to 1 in 80000 births and mortality is as high as 61-86 %.[2, 3]

Among the survivors, 80% has neurological sequelae.3 Clarke et al., [4]in the United States andTufnell[5] in the United Kingdom established a national registry for suspected AFE.

The diagnostic criteria are the presence of the following 4 signs or symptoms[3]:

1) Acute hypotension.
2) Acute hypoxia.
3) Coagulopathy or severe clinical haemorrhage in the absence of other explanations.
4) All of these occurring during labor caesarean delivery or dilatation and evacuation or within 30 minutes postpartum with no other explanation for the clinical findings.

It is often difficult to differentiate AFE from other types of thromboembolic events or other causes of cardiopulmonary collapse, although basic treatment principles are the same: oxygenating the patient, stabilizing hemodynamic parameters, and treating the underlying medical condition.

This case report includes the management of a patient with clinically suspected AFE.

2. Case Report

A 38 years old female gravida two para one was shifted to the labour room for labour induction with complain of pain abdomen and leaking PV with clear liquor. After around 30 minutes patient develops excessive sweating, not responding to verbal commands with vitals of BP 80/50 mm Hg, feeble pulse, Spo_2 76 % on room air, HR 82bpm (monitor), RBS 90mg/dl. Patient became confused and responded only to pain stimulus. She developed labored breathing, progressive bradycardia and suddenly went into cardiac arrest. There was perioral cyanosis and she developed tonic clonic seizure. Cardiopulmonary resuscitation was given immediately.

Atropine 0.6mg/ml and adrenaline 1: 10000 units were administered. Patient was intubated with ET tube 7.5 Cuff and was shifted to TICU. She was put on to a ventilator. Post resuscitation bp 150/100 mm Hg, PR 128, spo_2 98%. On examination multiple oozing of blood noted from the venous puncture site. ECG of the patient showed S1Q3T3 pattern and there was no urine output. A central venous catheter was placed at the right subclavian vein. Later, an IUD baby was delivered in ICU by obstetric surgeon. Gush of blood was noted after delivery of placenta. Immediately transfusion of packed cell and FFP was started.

After around 4 hours, bp dropped to 80/60mm Hg, PR100 /min, spo_2 100% with urine output 400ml. Norad (2amp plus 46 ml NS)[at]6 ml /hr was started and titrated accordingly. Injection calcium gluconate 10mg was also administered.

On the next day, postpartum day 1, patient was converted to T-piece, Norad was tapered and stopped. A total of four PRBCs and four FFP were given.

ABG showed ph.7.45, $pco_2$28, $po_2$189, HCO_3 19.3, spo_2 99.7%. Urine output of the patient decrease to nil over a period of two days. Echocardiography (bedside) showed mildly dilated right atrium with minimal to mild pericardial effusion.

On postpartum day 2, KFT was repeated and showed urea 94, creatinine 3.1, d-dimer 7.33. Chest x ray showed pulmonary edema, enlarged pulmonary artery.

On postpartum day 3, after assessing she was extubated however urine output for the last 24hours was 35ml only an was put on hemodialysis. After dialysis her creatinine deceased to 3.8, platelet 80000. She was orally allowed. IV fluids were tapered.

On postpartum day 4, patient complained of sob and cough. on examination bilateral crepts present, decrease breath sounds, chest x ray shows bilateral pleural effusion. Her test report reveals urea 134, creatinine 5.4, platelet 85000. Another setting of dialysis planned in view of fluid overload. A total of three episodes of haemodialysis was done. Urine output started to increase after day 11 of admission but went into diuresis phase. She was observed closely for her fluid status and overload for the next four days. Dietician consultation was done. Gradually her condition improved. Urine output was around 0.5ml/kg/hr and was shifted out of TICU. Patient's parameters at the time of shifting to the ward were Hb 9.7%. TLC 6.5, serum creatinine 4.7, urea 238, urine output 1.5L. On examination chest clear bilaterally, bp120/70, pr80, spo_2 98% on room air. Later she was discharge from ward after days of observation.

3. Discussion

Amniotic fluid embolism is a rare complication but has a high fatality rate, characterized by sudden cardiovascular collapse, dyspnea, or respiratory collapse and disseminated intravascular coagulopathy.[6]

This may occur in healthy women during pregnancy, labor, or following delivery. It can develop even after elective abortion, amniocentesis, cesarean delivery, or trauma. This condition is initiated by entry of amniotic fluid to the bloodstream of the mother, which leads to a serious reaction causing cardiopulmonary arrest and massive coagulopathy.[7, 8]

Because AFE is a diagnosis of exclusion, a precise case definition and criteria are difficult to establish and also other causes of maternal collapse should be ruled out. The causes of maternal collapse could be due to hemorrhagic shock, pulmonary embolism, anaphylaxis, septic shock, and aortic dissection.[9]

Diagnosis is based upon the signs and symptoms observed during the birth or procedure. In this case, the patient had no history of high blood pressure and she was always normotensive. She did not have a history of fever, and the investigation report also supported to rule out sepsis. She did not have postpartum hemorrhage, and she was not anemic prior to delivery, which helped to rule out hemorrhagic shock.[10]

The associated risk factors for AFE are age more than 35 years, multiparity, cesarean section, instrumental delivery, antepartum hemorrhage, eclampsia, labor induction, fetal distress, fetal death, and male baby.[11, 12, 13]

Few authors have proposed two clinical forms of AFE: typical and atypical. Typical or classic form has three phases: phase 1—respiratory and circulatory disorders, phase 2—coagulation disturbances, and phase 3—acute renal failure and acute respiratory distress syndrome (ARDS) leading to cardiopulmonary collapse.[10] In atypical form, cardiopulmonary collapse does not occur, but the first symptom is life-threatening hemorrhage due to DIC.[14]

The most significant diagnosis of AFE is made by findings at autopsy, which are limited to the lungs and clinical diagnostic criteria, and assisted by serum markers. Serum markers such as C3, C4, and C1 esterase inhibitors are reduced.[10]

The symptoms are usually sudden in onset. Acute dyspnea, agitation, sudden chills, sweating coughing, and anxiety are common premonitory symptoms. Labored breathing and tachypnea may occur. Diagnosis can be made by the following criteria [4, 15]:

1) Acute hypotension or cardiac arrest
2) Acute hypoxia
3) Coagulopathy or severe hemorrhage in the absence of other explanations
4) All of these occurring during labor, cesarean delivery, dilation, and evacuation or within 30 minute postpartum with no other explanation of findings

In our case, this woman developed cardiac arrest, cyanosis and tonic clonic seizure around 30 minutes after labour induction which fitted with the above-mentioned criteria of AFE. Echocardiography (bedside) taken on postpartum day one showed mildly dilated right atrium with minimal to mild pericardial effusion. Chest x ray taken on day two postpartum, showed pulmonary edema, enlarged pulmonary artery. A total of three episodes of haemodialysis was done in this patient.

Survival after AFE has improved significantly due to early recognition and management, and morbidity remains high with severe sequelae. Neurological impairment is the most common complication followed by renal failure, cardiac failure with left ventricular impairment, and arrhythmia have been reported.

In this case, the patient was given immediate CPR along with respiratory support. Keen attention was paid on nutrition and judicious use of fluids. Hence, survival and improved long term morbidities of AFE lies with early suspicion and diagnosis and aggressive management with resuscitation.

4. Conclusion

AFE is a rare but often fatal complication that may confront the anesthesiologist. Many of the initial findings are nonspecific and not life threatening, such as tachypnea, dyspnea, and tachycardia. However, severe complications such as cardiopulmonary collapse and bleeding coagulopathy can develop quickly, as seen in this case which was managed aggressively with resuscitation.

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